HUMAN TRYPANOSOMIASIS IN THE LUANGWA VALLEY, NORTHERN RHODESIA

BY

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I. PHYSICAL ASPECTS OF THE VALLEY

The Luangwa river rises in the hilly country near the junction of the Rhodesia-Nyasaland-Tanganyika Territory boundary at an altitude of from 5,500 to 5,900 feet and flows in a general south-westerly direction to its confluence with the Zambesi at Feira, 1,500 feet above sea level. On either side its valley is bounded by ranges of hills which run roughly parallel to the course of the river and which lie at distances varying from a few to as many as 40 or 50 miles from it. The country with which I am more immediately concerned in this report extends from Fundu at the southern end to the confluence of the Wira with the main river at the northern end, a distance of approximately 400 miles. Taking the average width of the valley to be 50 miles, the area thus included is at least 20,000 square miles. The Luangwa is fed by a large number of tributaries which enter it more or less at right-angles ; but whereas many of those on the right side are large and permanent streams, those on the eastern bank, with one exception, flow only in the rains, and then only assume perceptible volume when carrying flood water. In general the floor of the valley is level and is covered to a very large extent by ' mopani ' bush which becomes water-logged, if not actually flooded, during the rains, and in which the grass is short and of scanty growth. This mopani bush usually ends rather abruptly at some distance from the streams and is replaced by more or less open country covered by dense and luxuriant growths of grass. As it is only in these situations that the soil repays cultivation, the villages, which are small collections of from 20 to 200 inhabitants, are found strung along the courses of the larger streams. This is more particularly the case on the eastern

side of the Luangwa, as it is only in the beds of the larger streams that water can be obtained by digging in the dry season. It may be noted that at this time of the year the Luangwa itself dries up in stretches in the upper reaches.

The mean altitude of the area under discussion may be taken as about 2,300 to 2,500 feet, so that the meteorological conditions are much more tropical than in the other portions of Northern Rhodesia. At Nawalia, which is about centrally placed at an altitude of about 2,100 feet, the mean temperature varied from 67° F. in July to 87° F. in November, and the relative humidity from 31 per cent. in September and October to 78 per cent. in January. The rain-fall averages about 40 inches and is spread over the six months, November to April, though the bulk is precipitated during the first three months of the year.

II. DISTRIBUTION OF GAME AND FLY

Game is both extremely abundant and varied throughout the whole of the valley. In the rains it ranges through the whole of the area, but in the dry season, more particularly after the grass has been burnt (July to September), it tends to collect along the courses of the streams where the grazing is better than in the mopani and where alone a supply of water is assured. At this time of the year it is not uncommon to find a relatively large animal population in fairly close proximity to the villages, as many of the species, e.g., waterbuck, roan, eland and bushbuck, are fond of feeding in the old gardens. I think this point should be emphasised, as it was demonstrated at Nawalia that waterbuck and bushbuck, amongst others, were infected by *Trypanosoma rhodesiense*, the first-mentioned to the extent of between 17·8 per cent. and 25 per cent. of the total number examined. It may here be noted that in the immediate neighbourhood of some villages in Kambombo's country which have suffered severely from sleeping sickness, waterbuck are extremely plentiful. There are grounds, therefore, for regarding this species of buck with particular suspicion.

Fly. *Glossina morsitans* is to be found throughout the whole of the valley and has been observed to show much the same seasonal variation as the game, i.e., in the dry season it is abundant near

the streams and hence near the villages, but after the rains have commenced tends to become more uniformly scattered over the country as a whole. This peculiarity has been observed in other parts of Africa. Owing to the fact that the villages are, as a rule, built in clearings and are surrounded by gardens, it is unusual for the fly to invade them, though an occasional specimen may follow the natives in. If so, it soon disappears again.

III. HISTORY OF SLEEPING SICKNESS

Attention was first drawn to the occurrence of human trypanosomiasis in the valley by the diagnosis of the disease in several Europeans about the year 1909. Investigations commenced then and carried out in the succeeding years showed that it was scattered over the whole of the area though the number of cases found was comparatively small. They demonstrated further that the infection displayed no tendency to assume epidemic proportions. Writing at the end of 1912, the Principal Medical Officer stated : ' In the light of recent knowledge as to the presence of the necessary factors and apparently very suitable conditions for the production of an epidemic, it is difficult to understand why after four years there should be no evidence that such is likely to occur. It can be most reasonably concluded that there is some unknown but necessary factor wanting or some inhibitory influence present, i.e., that the disease is an old one and that there may be a certain immunity present which is limiting its spread.' This is in strict accordance with the native evidence, for those I have questioned have always consistently maintained that they have always known of the existence of the disease as far back as their memories carry, in some cases a matter of seventy years or so. They state that it cropped up only as isolated cases which were drastically disposed of in some localities. Amongst the Bawisa and Bansenga the terms ' Chilotera ' and ' Nyamakazi ' are used to denote the symptom-complex of fever, oedemata of the extremities, protuberant abdomen, diarrhoea and emaciation, all commonly observed in sleeping sickness, though, of course, they do not associate the disease with tsetse flies. My own belief coincides with that of the natives, that the disease is an old one and not of recent introduction.

IV. INCIDENCE OF THE DISEASE

The observations with which I am now about to deal are all, with few exceptions, derived from data collected by me during 1913 and the early part of 1914, and after the conclusion of the war, from 1920 onwards. In this work I have relied on gland palpation and puncture as the means of diagnosis and only departed from it in exceptional cases and for special reasons, e.g., where a native's illness appeared to be due clinically to sleeping sickness but no palpable glands were found. Under these conditions, I claim that the various sets of figures may be properly compared and that they give a real indication of the relative incidence of the disease in the different years and in the same localities. I do not claim, however, that they indicate the absolute incidence of the disease. While I am of the opinion that gland palpation and puncture is the quickest and only feasible method of examining large bodies of natives, and that the great majority of cases will be found by its employment, I admit that some cases, particularly those in the very early stages of the infection, before the lymphatic glands have hypertrophied and in which no other symptoms are present, will be missed.

As mentioned earlier, the disease is very widely distributed and has been found in the Petauke, Serenje, Fort Jameson, Lundazi, Mpika and Chinsali portions of the valley. Further it is, as a general rule, comparatively rare, as the following figures will demonstrate :—

Year examined	District	Natives examined	Cases	% infected
1913	Mpika	2,613	2	0·08
	Lundazi	13,100	9	0·07
	Chinsali	1,465	4	0·26
1914	Serenje	1,981	2	0·10
	Petauke	3,654	2	0·05
		23,113	19	0·08
1921	Lundazi, Part	3,723	1	0·03
	Mpika „	1,311	0	0·00
	Chinsali „	913	1	0·11
		5,947	2	0·03

As illustrating the general tendency for the disease to remain stationary over a period of years the following figures may be quoted. In every instance they are for the same villages in the respective years.

District	Year	Natives examined	Cases	Year	Natives examined	Cases
Lundazi	1913	2,812	0	1921	3,530	1
Mpika		1,233	0		1,261	0
Chinsali		776	3		913	1

A further example may be quoted from Dr. May's investigations in one particular area in the Petauke Sub-District where, in the three successive years 1910, 1911, and 1912 respectively, 7, 7, and 3 cases were found.

It is difficult, in the absence of a complete census and the registration of births and deaths, to estimate not alone the general death rate amongst these natives but also that more specifically due to sleeping sickness. The following calculations, however, may be given. At the end of 1912, Dr. May computed the adult death rate in a portion of the Petauke Sub-District to be 28 per 1,000 and the incidence of sleeping sickness to be 8 per 1,000, also amongst the adults only. In 1913 I made similar enquiries as carefully as was possible in the valley portion of the Mpika Sub-District and estimated the general death rate for the year 1912-1913, exclusive of accidents, to be 23·7 per 1,000, the adult rate to be 47·7 per 1,000 and the incidence of sleeping sickness to be 3 per 1,000 of the whole population seen. Speaking of the valley generally, I should be inclined to say that under ordinary circumstances the incidence of the disease is not in excess of 3 to 4 per 1,000 of the total population per annum, though of course it may be exceeded temporarily in those localities in which exacerbations of the infection occur (for example, the estimated death rate for 1920-21 of 25 per 1,000 in the countries of chiefs Tembwe and Kambombo).

A very striking feature of the infection is the extraordinarily sporadic manner in which it is found. Not only are the cases found in villages widely separated but they are also usually found occurring singly, and cases in the same village may be separated by an interval of years. Examples of this are given in the following tables, and it should be noted that approximately the same number of natives were examined on the various occasions.

Village	Wallace, 1912	Kinghorn, 1913
Mumamba	1	0
Daroba	1	0
Chuni	0	1
Mkasanga	0	1
Chombero	0	1
Kundawamawe	0	1
Temba	0	1
Chinyondo	0	1
Luchenga	0	1
Kampuzunga	0	1
Mulumgu	0	1

Village	1920	1921	1922	1924	1925
Tembwe Virizi	0	2	1	0	0
Katangalika	0	1	0	0	0
Ng'anjo	1	0	0	0	0
Mwimba	0	1	0	0	0
Kajumba...	1	1	0	1	0
Kambombo	0	1	0	0	0
Chizonde...	0	1	0	0	0
Kambwiri	1	0	0	0	0
Kazembe...	0	0	1	0	0
Dungulungu	0	1	0	0	0
Chama	1	1	0	0	0
Kawanda...	1	1	1	0	0
Kapalakonje	1	0	0	0	0
Chitimbe...	1	1	1	0	0
Nyika	2	1	0	0	0
Luambo	2	0	1	0	0
Chileta	0	0	1	0	0
Hunga	1	2	0	0	0
Buli	1	0	0	0	0
Chitukula	1	0	0	0	0
Chiruarua	2	0	0	0	0
Zowole	1	0	0	0	0
Mtonya	1	0	0	0	0
Mkunguwe	1	0	0	0	0
Luchenga	0	0	2	0	1
Kapotwe	0	1	0	0	0
Makondola	1	0	2	0	0
Kakuni	0	0	2	0	0
Marunga	1	0	0	0	0

How is this peculiar distribution and incidence to be explained ? As is well known, some observers, chiefly the Germans, maintain that there are two distinct trypanosomes, *brucei* and *rhodesiense*, existing side by side in tropical Africa, which are indistinguishable except for the fact that *T. rhodesiense* is capable of infecting man while *T. brucei* is not, but is restricted to game and stock. The other school, chiefly British, maintain that the two trypanosomes in question are identical ; that it is essentially a parasite of game ; and that man is ordinarily resistant to infection, though this may occur. Exactly what conditions are necessary before man does become infected are uncertain. The chief arguments of those who favour the non-identity hypothesis are : (1) Dr. Taute's experiments, and (2) the geographical argument, that in many localities where *T. brucei* is found cases of sleeping sickness have never been diagnosed. With reference to the first of these, I think the experiments may be held quite permissibly to prove the truth of the contention that man is naturally resistant to infection by *T. brucei vel rhodesiense* and that, in any event, they are not sufficiently extensive to prove indisputably the truth of the negative statement that the human and game trypanosomes are not identical. As regards the geographical argument, I am not aware that the localities usually cited have been thoroughly and repeatedly examined over a period of years, and that, at the same time, such important factors as the abundance of fly and game, the percentages of both harbouring *T. brucei, sensu strictu,* and the closeness of contact existing between them and the native population have been taken into consideration. Without departing from the confines of Northern Rhodesia, however, it appears to me to be a difficult matter to explain, on the assumption that human cases of the disease are invariably due to a specific human as distinct from the ordinary game trypanosome, the occurrence of one European and two or three native cases in the western part of the Serenje sub-district with an interval of years between them, more particularly as the examinations of the suspected area carried out by Dr. Masters prior to 1912, by Dr. Ellacombe in 1912, and again by Dr. Powell in 1920, gave negative results as far as the indigenous population was concerned. And further, it is peculiar that in the other area in this country in which the disease has been found

to occur much as it does in the Luangwa valley the same conditions of an abundance of game and fly co-exist in close contact with the natives. I refer to the focus in the south-western corner of the Ndola sub-district. Prof. Kleine, one of the protagonists of the non-identity theory, admitted in conversation that the local game was susceptible to infection by *T. rhodesiense, sensu strictu*, and this being the case it follows that it would only be a matter of time until this parasite was widely distributed amongst the fauna of the valley and one would then expect to find human cases to be both more numerous and more uniformly spread over the country as a whole. As pointed out above, there is a concentration of both the game and the fly in the vicinity of the villages in the dry season, and while a certain amount of evidence exists to show that the risks of infection are then greater it is not extensive enough to permit of any dogmatic statement on the point. I believe, personally, that the sporadic appearance and erratic distribution of the infection as it is generally seen and has been seen since 1909, with no tendency to assume epidemic proportions, is more satisfactorily explained by the theory that the human and game trypanosomes are one and the same parasite and that man is ordinarily resistant to infection by it than by the theory that the human and game trypanosomes are distinct entities.

While the normal incidence of the infection is as shown above, exacerbations have been observed from time to time in localized areas of the valley, but these, after the lapse of a few years, have always ended spontaneously and the disease has then reverted to what may be termed its equilibrium. The most pronounced of these has been the one which started early in 1918, in the contiguous territories of the three chiefs Chikwa, Tembwe and Kambombo towards the Northern end of the Lundazi sub-district. Of the three, Kambombo suffered most. Thus writing in May, 1920, the Native Commissioner, Lundazi, reported that since the commencement of this outbreak some 131 deaths had occurred to date from what appeared to be sleeping sickness, amongst an adult population estimated at 1,455 on March 31st, 1920, in the villages under that chief. It may be noted that at the end of 1917, or beginning of 1918, plague appeared in this same area and was not stamped out until 1919. This coincidence was probably quite fortuitous except that many of the

villages were burnt and the natives forced to build temporary quarters in the bush. This, of course, brought them into closer and more continuous contact with fly than is ordinarily the rule, and to some extent an increase in the number of cases of sleeping sickness may be ascribed to this cause, but it cannot have been of general application, as those villages which suffered most severely from sleeping sickness were not destroyed. In 1920, and the succeeding years, I examined and re-examined the natives in this particular area with the following results :—

Year	Natives examined	Cases	% infection
1920	5,756	25	0·43
1921	5,634	18	0·32
1922	5,317	9	0·17
1924	4,347	1	0·02
1925	4,301	2	0·04
Compare 1913	5,122	3	0·06

Or if we consider the three sets of natives separately we have the following percentages of infection in the various years :—

Year	Percentage Infection		
	Chikwa	Tembwe	Kambombo
1920	0·25	0·73	0·36
1921	0·15	0·27	0·57
1922	0·22	0·12	0·15
1924	0·00	0·00	0·06
1925	0·07	0·00	0·06

It should be noted that these percentages only represent the incidence of the disease at the actual time of examination, and, while under ' normal ' conditions they might afford an approximate idea of its frequency, they are much too low for ' Epidemic ' conditions. Thus in 1921, by tracing the causes of deaths in the interval between that and the former visit, I estimated that the then rate of infection was about 2·5 per cent. of the whole population per annum. I am not fully satisfied that the 1924 figures give anything like a true indication of the percentage of infection in that year, as I saw about a thousand fewer natives than in former trips, and amongst those who evaded examination cases may have existed ; but it may be noted that the deaths between 1922 and 1924, which could be attributed to sleeping sickness were comparatively few ; hence it seems reasonable to conclude that this localized ' semi-epidemic ' is behaving like the others and that the infection is becoming stabilised again. That this conclusion is essentially true is borne out by the results of the trip I have just finished through this area. As will be seen above, 4,301 natives were seen and 2 cases of the disease diagnosed, a percentage of 0·04. In the six months which elapsed between my 1924 and 1925 trips some nine deaths, which might possibly have been due to sleeping sickness, occurred, so that it would appear that the annual incidence of the disease in this particular area is now not in excess of 5 per 1,000 of the total population.

I am of the opinion that these epidemics are due largely to the superimposition of a man-fly-man cycle of transmission of the parasite on the more ordinary game-fly-man cycle and the occurrence of this is often hastened by some of the habits of these natives. For instance, cases may be carried from one village to another owing to the custom of returning to the original home when a native falls sick. Again, during the rains the villages split up, each family living in the middle of its gardens in order to protect them from the depredations of monkeys and elephants, and cases of infection in man and wife and parent and child have been observed under these conditions. Further, the habit of growing crops between the huts in the villages themselves increases the liability of their invasion by fly with the consequent danger of their becoming infected should a case of sleeping sickness exist.

V. THE DISEASE IN HUMAN BEINGS

The incubation period is short, probably between one and two weeks. At first the only symptom is fever with its concomitants, followed by enlargement of the lymphatic glands, particularly in the basal portion of the posterior neck triangles, progressive emaciation, oedemata of the extremities and face, protuberant abdomen, diarrhoea, anaemia, muscular tremors, inco-ordination, mental hebetude and somnolence deepening into coma. In untreated cases, death is the inevitable result and the whole duration of the infection is short, on the average from about three to six months. An occasional case may live for ten or twelve months, though this is very exceptional, and only one, in my experience, has ever exceeded this period.

This native, Chimwila, was found to be infected on the 18th November, 1920, and then gave a history of having been ill only one month, complaining of headache. This was probably an underestimate as his neck glands were markedly enlarged. He was seen again on the 9th May, 1921, and appeared to be in perfect health saying himself that he was not sick. His neck glands were now smaller though the juice still contained parasites. His condition remained the same on the 11th March, 1922, and trypanosomes were still found in the gland juice. He was then sent to Prof. Kleine for treatment with Bayer 205, i.e., sixteen months after he had been found to be infected and probably eighteen at least after he contracted the disease. Prof. Kleine states in one of his reports that Chimwila appeared to be physically in good health on his arrival but that some mental dullness was noticed. After receiving treatment he returned to his village later in 1922 and was apparently quite well when last seen in March, 1925. No glands were palpable and no parasites were found in the blood.

The disease is very decidedly one of adolescence and adult life. I have never seen a case in a young child, and only comparatively rarely under the age of about fifteen. This is well brought out in the following tables from two independent sources, and in view of the great difficulty of estimating at all accurately the ages of natives and the part the personal equation must play in such estimations, the general agreement of the two sets of figures is very striking.

Age	Kinghorn		Fischer	
	Cases	%	Cases	%
6-15	5	6·66	1	3·57
16-25	20	26·66	9	32·14
26-35	27	36·00	11	39·28
36-45	17	26·66	5	17·85
46-55	5	6·66	1	3·57
Over 55	1	1·33	1	3·57
	75	99·97	28	99·98

It will be noticed from these tables that there is a steadily increasing susceptibility to infection from the earlier ages to about the 35th year and that thereafter the decline in susceptibility is as equally and steadily marked; further, that from 85 to 89 per cent. of all the cases occur in the age group 16 to 45. These remarks apply with equal force to the two sexes considered separately, as shown below.

Age	Males		Females	
	Cases	%	Cases	%
6-15	4	8·88	1	3·33
16-25	11	24·44	9	30·00
26-35	15	33·33	12	40·00
36-45	12	26·66	5	16·66
46-55	3	6·66	2	6·66
Over 55	0	0·00	1	3·33
	45	99·97	30	99·98

This table also brings out the fact that the disease is commoner in men than in women, the proportion being as 3 : 2, and this is corroborated by an analysis of Dr. Fischer's cases, which gives a proportion of $3\frac{1}{2}$ males to 2 females. Probably the greater number of male cases may be accounted for on the basis that the men, on the whole, spend more time in the bush, hunting, collecting honey, poles for building, reeds and other material for mat and basket-making, and so on, than the women do in their daily trips to collect firewood. It is more difficult to explain why, approximately after the age of puberty is passed, the susceptibility to infection should increase so perceptibly and increase in an ascending curve to the age of thirty-five and then decrease steadily. Babies and very young children are always carried by their mothers in a calico or sling and are thus fairly effectively protected, but the older children who also usually accompany their mothers wherever they go are naked and unprotected. The older children, aged from ten to twelve onwards, are, at best, very scantily dressed and have to assist their parents in the various duties outlined above. They are thus frequently exposed to the risk of infection and yet it is only very rarely that this occurs. It may be argued that the discrepancies in the age-incidence of the disease are more apparent than real and that if the actual age distribution of the whole population could be plotted, these would become obvious, but in the absence of an accurate census this is impossible. I am, however, inclined to doubt this. Certainly as between children and adults it does not apply, as it may be said generally that the number of children in a village will about equal the number of adults. Thus an analysis of the two groups in the villages under Chikwa, Tembwe and Kambombo, gives 2,741 children to 3,015 adults. I am convinced that the varying rates of infection amongst the different age-groups cannot be explained wholly and satisfactorily on the assumption that they are in direct ratio to the risks of infection run by the respective groups, though it is difficult to suggest other factors which may influence the occurrence of the disease in the light of our present knowledge. It may here be pointed out that hunger with its consequent lowering of vitality does not increase susceptibility to infection. In this area the 1923-24 crops were bad, with the result that, towards the end of 1924, acute hunger,

amounting in some villages to actual starvation, became apparent, and several deaths from this cause were reported. The results were particularly noticeable in Chikwa's country, where all of the natives showed emaciation varying from slight to extreme, yet only one case of sleeping sickness was found, and only five deaths from what may have been sleeping sickness were reported amongst 1,400 natives.

There is no evidence that acquired immunity to the infection is found in man though, as stated above, I am of the opinion that he is naturally very resistant to it.

VI. TREATMENT WITH 'BAYER 205'

Early in 1922, a commission composed of Prof. Kleine and Dr. Fischer came out to this country to try the effects of the drug known as 'Bayer 205' on human trypanosomiasis and a camp was established in the Luangwa valley at Ndombo, about thirty-five miles east of Mpika. In all, 38 cases were treated, and of these one man went later to Southern Rhodesia and has not been traced, five are still alive, and the remaining 32 are dead. Five of the deaths occurred at the Commission's camp from intercurrent affections and the other 27 in the villages at varying periods after the parents had returned there on leaving Ndombo in October, 1922. The following is the complete list :—

Name	Sex	Age	Date of death	Duration of life after treatment	Remarks
1. Pitala	M	25	February, 1924	16 months	
2. Yatula	F	30	—	—	Alive
3. Chimwila	M	35	—	—	Alive
4. Chizilicho	M	35	March, 1924	17 months	
5. Isake	M	15	July, 1923	9 ,,	
6. Chilikupata	M	25	April, 1924	17 ,,	
7. Mofati...	M	35	?	?	Untraced
8. Lamek	M	16	—	—	Alive
9. Samuel	M	16	February, 1924	16 months	
10. Tadeyu	M	27	February, 1924	16 months	

Name	Sex	Age	Date of death	Duration of life after treatment	Remarks
11. Kajawa	M	38	—	—	Alive
12. Vioka	F	22	October, 1923	12 months	
13. Mateyo	M	35	February, 1923	4 months	
14. Chifundulwa	M	60	—	—	Alive
15. Lasalu...	M	25	February, 1924	16 months	
16. Vilauli	M	38	July, 1924	19 months	
17. Malita	F	22	November, 1923	13 ,,	
18. Mwipi	M	12	December, 1923	14 ,,	
19. Murere	F	45	October, 1923	12 ,,	
20. Nderema	F	40	October, 1923	12 ,,	
21. Ntanda	F	27	October, 1923	12 ,,	
22. Yumba	F	45	November, 1923	13 ,,	
23. Ndabeya	F	54	April, 1923	6 ,,	
24. Mage	F	26	February, 1924	16 ,,	
25. Marata	M	28	July, 1923	9 ,,	
26. Mapulanga	M	35	February, 1923	5 ,,	
27. Kabrieni	M	18	November, 1923	13 ,,	
28. Chikoti	M	35	October, 1923	12 ,,	
29. Kamchepa	F	32	October, 1923	12 ,,	
30. Zakeyo...	M	35	October, 1923	12 ,,	
31. Wayilipa	F	35	October, 1923	12 ,,	
32. Sabeta	F	20	October, 1923		Died on road home
33. Thomas	M	17	February, 1924	16 months	
34. Thomas	M				
35. Ngoza...	F				
36. Chiweza	M	died at Ndomba from intercurrent disease			
37. Tepatepa	M				
38. Puntayila	M				

As will be noticed, the cases were of both sexes and of all ages and it may be added that they were in various stages of the disease from early to late. With regard to the cases which died in the villages, the native evidence is that at varying periods after they had returned they started to become ill again and presented the ordinary symptoms of sleeping sickness, e.g., progressive emaciation, oedemata, and so on. This applies equally to the eight cases which died in October, 1923, but there is a possibility that in these death was hastened by influenza of which an epidemic swept through the valley in August and September of that year. This reappearance of symptoms must be regarded as due to relapses of the original, and not to fresh infection. In the treatment of sleeping sickness the greatest drawback to success is the difficulty of attacking effectively the parasites in the cerebro-spinal fluid, and evidence of this with 'Bayer 205' is found in the reports of the German Commission. Thus it is stated that of twenty-one patients examined by lumbar puncture before their discharge from the Ndombo Camp in October, 1922, eight were found to harbour trypanosomes. Under such circumstances it would only be a matter of time until the trypanosomes re-invaded the blood stream and set up symptoms of the disease. The only definite result claimed by the Commission for 'Bayer 205' is that by its use it is possible to sterilise the blood for a long period even in those cases which are not clinically cured ; and while this claim seems to be established I question, in view of the demonstrated fact that the bulk of the cases did ultimately relapse, whether there are any substantial grounds for stating that ' if in districts infected with sleeping sickness all suspected natives receive treatment . . . the source of infection for the tsetses will gradually disappear and in time the disease must die.' It is assumed that man is the only reservoir and that the game is negligible. This I cannot admit.

The routine method of treatment adopted by the Commission was three subcutaneous or intravenous injections of 1·2 gm. in normal saline at intervals of ten and eighteen days, though those cases in which the parasites persisted in the blood and cerebro-spinal fluid received a fourth and fifth injection. It is apparent, therefore, that a dosage of from 3·6 to 6 grammes is insufficient to cure the majority of cases of *T. rhodesiense* infections in natives. Better

results might, of course, follow the adoption of an increased dosage up to 10 or 12 grammes as has been advised by some experienced workers, but at the price of 6/6 a gramme it is questionable whether the expenditure of £3/5/– to £4 per head on drugs alone would be legitimate, in the light of our present knowledge, on any very wide scale, more particularly as reports from the Congo would indicate that in tryparsamide we possess an equally, if not more, efficacious drug which possesses the advantage of being cheaper. It is of interest to note that in these reports it is said that the results of treatment there with 'Bayer 205' had been 'frankly disappointing.'

I saw and examined the five cases which are still alive in August, 1924, and again last month. All of them appeared to be in perfect health, presented no signs or symptoms of sleeping sickness, and showed no parasites in the blood stream. It was not possible to perform lumbar puncture. I believe, therefore, that these natives may now be regarded as being definite cures. Disregarding the five cases which died from intercurrent disease and the one which has not been traced, this represents a percentage of 15·6 cured. In view of the fact that all previous methods of treatment for the Rhodesian type of the disease have been failures, this must be admitted to be a considerable advance, but these results do not justify the very optimistic claims which are still being made for this drug.

VII. PROPHYLAXIS

It is a definite fact, which must be recognised, that no active assistance can be expected from these natives for any measures designed either to combat a specific infection, or generally to improve the sanitation of the villages. For some years movement into and out of the valley was prohibited, and even though the sleeping sickness regulations have fallen into comparative desuetude very few Europeans travel there now. Thus to a marked degree the natives have remained under the influence of their ancient tribal beliefs and treat European ideas of the etiology of disease and the methods to be adopted in treating them with undoubted, if unexpressed, disbelief. The general attitude, therefore, becomes one of passive resistance which can only be overcome by a certain

amount of compulsion, and when force has to be employed the results are usually unsatisfactory. Not only does the native revert to his own ideas and habits as soon as he thinks he can safely do so, but there is also a tendency to evade actively the application of disagreeable rules and regulations by running away and hiding as soon as an official appears in his district. Of this I have had personal experience. This becomes more pronounced when the results of European regulations and treatment are unsatisfactory, as it must be admitted they have so far been with particular reference to sleeping sickness. My experience is that the few cures are overlooked and attention is concentrated on the failures, and that indeed the natives really believe that the deaths have been caused by the use of hypodermic needles. In time, and with the increase of education, this general attitude may be modified but it will necessarily be a very slow process, and in the interval it is not apparent that much can be done. Something might be done to hasten this, by arrangement with the various missions having schools in the valley, if the teachers were given instruction in the essential facts of the etiology of sleeping sickness and other infections, told to explain these facts to their classes at frequent intervals, and to urge patients to go to hospital for treatment. No immediate results could be expected in view of the ingrained instinct of the native to return to his home as soon as he falls sick, and in view of the widespread aversion to going into hospital. I think, however, it would be a step in the right direction.

In view of the facts, which I think have been brought out earlier, that sleeping sickness ordinarily is one of the less important causes of death in the valley and that in general it occurs only in widely-scattered, sporadic cases, I am inclined to doubt whether it is incumbent on the Government to provide special facilities for treatment beyond those which already exist in the various hospitals. When the enormous area of the valley is recalled it is obvious that to do so would entail, if success is to be obtained, the appointment of at least three special medical officers with hospital and staffs, and the expense of this would, of course, be very large. Success could only be anticipated if the theory is true that *T. rhodesiense* is a parasite of human beings alone, that man is the sole reservoir and that the game plays no part in the perpetuation of the infection.

If so, then it appears to me that the obligation to deal with the disease by special measures is greatly strengthened. If, however, it is correct that the game and human parasites are identical, or alternatively that the game may act as a reservoir for a specific human trypanosome, then success in combating sleeping sickness can only be assured by simultaneously segregating and treating all the human cases of the disease and killing off the whole animal fauna. This is not a practicable proposition in the valley.

In the event of the occurrence of one of the localized epidemics it would, I think, be advisable to institute local treatment, as I believe that in these man does play a part as a direct reservoir of infection and it would be a matter of some importance to break the man-fly-man cycle.

I also think that it would be advisable to keep on the statute book the Sleeping Sickness Regulations, not with any idea of interfering with the natives, but chiefly for the power they confer of regulating the movements of Europeans. If all the regulations were rescinded, there would probably be an influx of professional hunters in pursuit of elephant, and the possible occurrence of cases amongst them might entail an unnecessary expense on the Government.

Lundazi, N. Rhodesia,
April 10, 1925.